CASE REPORT

RUPTURE OF THE PULMONARY ALVEOLI DURING GENERAL ANESTHESIA[1, 2]

Ronald Miller, DDS[3] and Frank Bongiorno, MD
USAF DC, Department of Oral Surgery
USAF Hospital, Yokota, Japan
USAF, MC, Department of Surgery
USAF Hospital, Yokota, Japan

SUMMARY

The rupture of pulmonary alveoli during the induction of general anesthesia is a significant complication of general anesthesia that has been seldom reported in our literature. This article documents this occurrence in a patient scheduled for elective orthognathic surgery. The diagnosis and treatment are discussed.

Excessive airway pressure in the intubated patient can cause a rupture of the alveolus of the lungs. This is a unique anesthetic complication which has been rarely described in the literature.[1-6]

The following is a case involving an alveolar rupture secondary to excessive ventilatory pressure immediately following the induction of general anesthesia and the placement of an endotracheal tube and a nasal gastric tube.

REPORT OF CASE

A 5'8", 135 lb, 26 year-old white female was admitted for elective orthognathic surgery under general anesthesia. Significant medical history findings included a 10 pack-year cigarette usage and two uncomplicated natural deliveries in 1978 and 1982. The rest of the physical findings, pre-operative blood workup, urinalysis and chest radiograph were noncontributory.

After induction of general anesthesia with intravenous thiopental sodium (Pentothal, 350 mg), tubocurarine chloride (3 mg) and succinylcholine chloride (Anectine, 80 mg), a nasal endotracheal intubation was performed under direct vision with a laryngoscope. After the tube was secured to the head, 4 liters per minute of N_2O, 2 liters per minute of O_2 and a 2% flow of enflurane (Ethrane) was begun. The patient was then placed on a ventilator which was set to provide 700 cc per ventilation at a rate of 10 ventilations per minute and a maximum inflation pressure of 100 mm of water. Pulmonary compliance appeared to be within normal limits.

Following placement of a nasal gastric tube, intermittent premature ventricular contractions (PVC's) were noted on the electrocardiagram. Mechanical ventilation was terminated and the patient was manually hyperventilated. This resulted in resolution of the cardiac dysrhythmia. However, a decrease in compliance was noted. Auscultation of the lungs was unremarkable, but subcutaneous emphysema was noticed extending from the clavicles to the infraorbital areas bilaterally.

Also noted at this time was an area of cutaneous venous congestion on the upper chest which readily blanched on digital pressure. During a second auscultation, breath sounds were more distant. The EKG pattern revealed a normal sinus rhythm with a rate of about 70. The anesthetic gases were discontinued and ventilation was supported with 100% oxygen. Surgery was cancelled, and a portable chest radiograph and arterial blood gases were ordered. At this time the patient was experiencing mild dyspnea, and her nail beds were cyanotic.

The chest radiograph showed an 80% pneumothorax on the left side, a pneumomediastinum and pneumoperitoneum (Figure 1). The arterial blood gases showed a pH of 7.43, PO_2 of 381, PCO_2 of 42, O_2 saturation of 100%, and base excess of +3.4 after hyperventilation with 100% O_2.

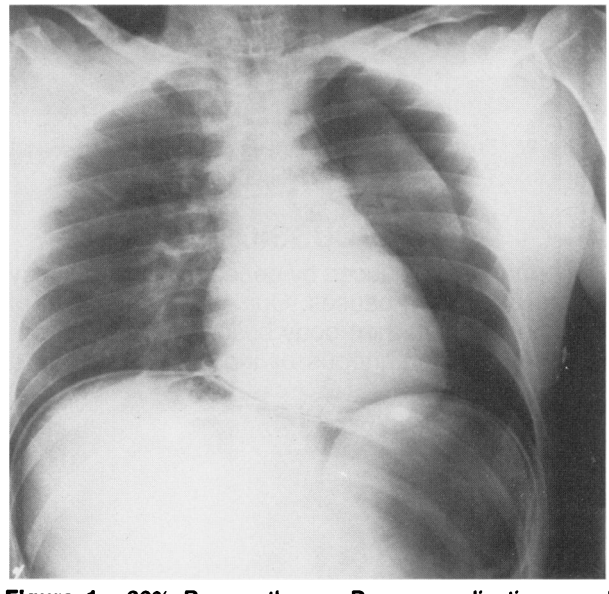

Figure 1. 80% Pneumothorax, Pneumomediastinum and Pneumoperitoneum.

[1]Accepted for publication October 12, 1982.

[2]The opinions contained in this article are those of the authors and are not to be considered as official or as reflecting the views of the Department of Defense or the United States Air Force.

[3]Dr. Miller is a LtCol, USAF DC, Department of Oral Surgery and Dr. Bongiorno is a Major, USAF, MC, Department of Surgery, at Yokota USAF Hospital, Japan, APO 96328. Requests for reprints should be sent to Dr. Miller.

A chest tube was placed at the third intercostal space in the mid clavicular line and attached to a pleurevac set at −20 cm water pressure. The patient improved clinically and after 2 hours of observation a new radiograph showed resolution of the pneumothorax and pneumomediastinum, with persistence of the pneumoperitoneum. The patient was extubated, and her post-operative recovery was uneventful. Because she had no throat or neck symptoms, she began feeding 24 hours later. The chest tube was removed 48 hours later. A radiograph at that time showed fully expanded lungs, resolution of the pneumomediastinum, and some residual pneumoperitoneum (Figure 2). The patient was followed on an outpatient basis and has continued to do well. The ventilator was checked for malfunctions and no mechanical problems were found.

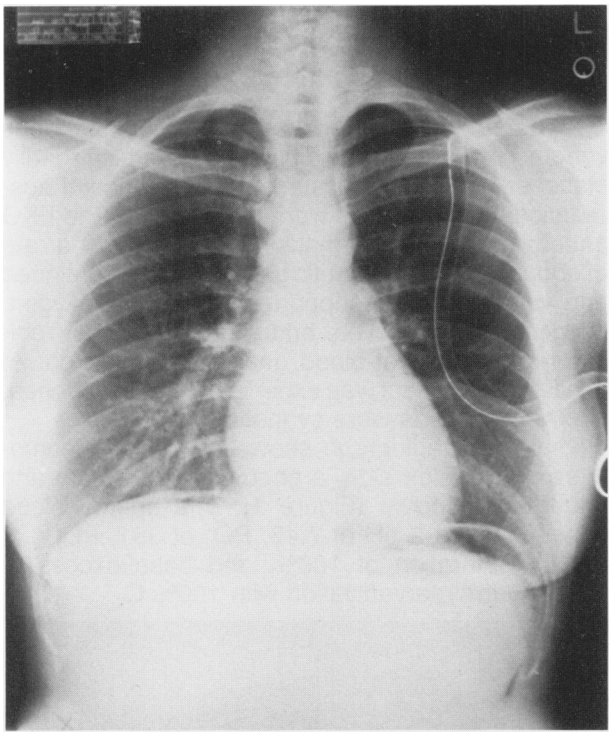

Figure 2. Pneumothorax and Pneumomediastinum resolved. Pneumoperitoneum decreased but still present.

DISCUSSION

Pneumothorax induced by general anesthesia may have a number of causes. Our patient, because of her asthenic (tall, thin) body build, would be a candidate for a spontaneous or induced rupture of a congenital "bleb". In this form of pneumothorax, the preeminent feature is the air in the thoracic cavity. Other clinical findings such as pneumomediastinum and subcutaneous emphysema are secondary features. In this situation, the ruptured bleb can form a check valve mechanism allowing air into the cavity but not out, leading to the rapid induction of a tension pneumothorax with the positive pressure ventilation of anesthesia.

More commonly a pneumothorax during anesthesia is caused by the overdistension of the lungs by positive-pressure ventilation. Air may be forced out the alveolar spaces and may dissect along perivascular and peribronchial sheaths to the mediastinum. More pressure and air propels this mediastinal air proximally to the cervical fascia and distally to the abdominal cavity. This results in subcutaneous emphysema and pneumoperitoneum. Air may also, but not inevitably, dissect into the plural space, and produce pneumothorax indirectly. This is in contrast to the direct instillation of air through an open bleb.

Though the pneumothorax derived from excessive pressure during anesthesia would appear to be a potential disaster, the cases reported in the literature have all had successful outcomes. Undoubtedly, the rapid availability and interpretation of chest radiographs coupled with swift and accurate chest tube placement has contributed to the lack of mortality.

Additionally, the great majority of cases are due to an overextension mechanism which has the preeminent sign of massive suscutaneous emphysema rather than the rapidly advancing tension of a ruptured bleb with an open check valve.

Our case seems to fit this category since the patient did not develop a pneumothorax until well after subcutaneous emphysema was noticed.

REFERENCES

1. Newton NI, Adams AP: Excessive airway pressure during anesthesia: Hazards, effects and prevention. Anaesthesia 33.689, 1978.
2. Sears BE, Bocar ND: Pneumothorax resulting from a closed ventilator port. Anesthesiology 47:311, 1977.
3. Barale F. Boilot A: Preoperative pneumothorax. Ann Anesthesiol Fr 18:619, 1977.
4. Rastogi PN, Writh JE: Bilateral tension pneumothorax under anesthesia. Anaesthesia 24:249, 1969.
5. Chiles DG, Mannheimer WN, Miller FE: Pneumothorax, pneumomediastinum and massive subcutaneous emphysema after general anesthetic. J. Oral Surg 28:845, 1970.
6. Sandor GKB, and Tolas, A: Spontaneous Tension Pneumothorax following outpatient general anesthesia, J. Oral and Maxillofacial Surgery 40:594-6, 1982.